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Summer Preceptorships

SUMMER PRECEPTORSHIPS for medical students are nothing new. But what is new is the need for many more students than before to garner some dollars during their summers to help them meet some of the ever-rising costs of getting a medical education. Elsewhere in this issue is an account by a second-year medical student of his most worthwhile experience as a summer preceptee, and of his gratitude for being taken into the home of his preceptor and so being able to save quite a few extra dollars for his needs in the coming academic year.

It is evident that both student and preceptor gained much from this experience. But beyond this, it is an expression of one of the oldest and grandest things in the Hippocratic tradition of medicine, ". . . to teach them this art, if they shall wish to learn it, without fee or stipulation; and that by precept, lecture, and every other mode of instruction, I will impart a knowledge of the art . . . to the disciples who have enrolled themselves and agreed to the rules of the profession. . . ."

Things are surely quite different here and now than they were on the island of Cos in the late 5th century BC. But the need to pass the art from preceptor to preceptee, from a practitioner who has mastered it to a student who must learn it, has not changed, and the ancient preceptor-preceptee relationship remains a valuable experience for both. Summer preceptorships are to be encouraged, and the dollars, which are more important now than they were 2,500 years ago, should somehow be found for those who need them. These experiences add an important dimension to the education of a young physician—a dimension that practicing physicians are uniquely able to give.

—MSMW

Reye's Syndrome and Ondine's Curse

THE PULMONARY CONFERENCE REPORT from Stanford in this issue includes a most elegant, comprehensive discussion, by Eugene Robin, of various pathophysiologic processes inducing hypoventilation. An almost bewildering variety of syndromes are present in patients with neurologic, airway, pulmonary, hepatic and other disease processes. I wish I could offer the reader the broadly sweeping brush of simplification, but in fact, what little I can add is further complexity.

A minor omission from the discussion and Dr. Robin's Table 1 is the role of the carotid and aortic body peripheral chemoreceptors in their response to arterial hypoxia and acidemia. More common than direct lesions of carbon dioxide chemoreceptors is loss of ventilatory drive usually transmitted by the IX cranial nerve from these glomera. Carotid bodies may be inadvertently denervated during carotid endarterectomy,[1] or deliberately excised to ameliorate asthma.[2] Chronic hypoxia gradually "blunts" its own response, and about 10 percent of the normal population is virtually unresponsive to hypoxia, suggesting that such defects are benign. More serious is damage to IX nerve roots by meningitis or movement of the brain stem. The absence of gag and laryngeal reflexes in the child subject of this report suggests IX nerve involvement, and resembles the syndrome Mitchell and I observed in a patient whose IX nerve roots were avulsed by a surgical attempt to relieve cervical cord traction

pain of platybasia.[3] She probably also had X nerve root damage, and when neglected for a few minutes, would forget to breathe, a victim of Ondine's curse. Usually, the total loss of peripheral chemoreceptor drive does not lead to respiratory failure like that seen in this child, but does induce a 15 percent rise in $Paco_2$ to about 46 mm of mercury, and converts the effect of hypoxia from stimulation to depression of ventilation.

The medullary carbon dioxide chemoreceptors are apparently nerve endings lying on or near the surface of the ventral medulla,[4] where one might expect them to be easily attacked by infection or toxins in cerebrospinal fluid, yet as far as I know no one has obtained pathologic evidence of such a lesion producing Ondine's curse or respiratory failure. The site of lesions inducing hypoventilation seems usually to involve the nuclei of the glossopharyngeal and vagal nerves, where the real business of regulating automatic ventilation is carried on. As Robin points out, pathways of voluntary ventilatory control are sufficiently remote from these nuclei to be spared in some patients who have lost automatic drive.

The normal balance of carbon dioxide and oxygen related drives is heavily tilted to carbon dioxide. About 85 percent of our resting or sleeping ventilation depends on stimuli from hydrogen ions in the extracellular fluid of these medullary chemoreceptors, as controlled by arterial carbon dioxide pressure ($Paco_2$)—provided that arterial oxygen presure (Pao_2) is greater than 80 mm of mercury. When Pao_2 falls to 40 mm of mercury (75 percent saturation of hemoglobin), the tilt is reversed, and resting ventilation is multiplied by some three to five fold (at constant $Paco_2$). I use the term multiplied because both human and animal experiments indicate that the effect of hypoxia multiplies rather than adds to carbon dioxide drive, or in graphic terms, hypoxia steepens the slope of the ventilatory response to carbon dioxide. Over the range we feel safe in studying in man this seems to result in a curious phenomenon: This strong response to hypoxia is essentially eliminated by a small fall of $Paco_2$ (about 5 mm of mercury) to what is called the apneic threshold. If a normoxic normal subject is sedated (or anesthetized), and is mechanically hyperventilated to a $Paco_2$ below his threshold for a few minutes, he subsequently will show either apnea or "ataxic" hypoventilation until $Paco_2$ climbs back to this threshold.[5] Awake subjects usually have too much overriding "wakeful" drive to manifest apnea, as noted by Robin with respect to the Plum test. The upshot of this carbon dioxide-oxygen interaction is that acute hypoxia strongly stimulates ventilation, which soon lowers $Paco_2$, which then turns off the stimulus. We reach a new steady state, for example at a Pao_2 of 40 mm of mercury (as if suddenly transported to 14,000 feet altitude), after $Paco_2$ falls about 4 mm of mercury, canceling four fifths of the hypoxic drive, leaving ventilation only marginally increased (by the fraction 40/36, or 11 percent in this example).

It remains uncertain whether very severe hypoxia can directly stimulate breathing, bypassing this carbon dioxide multiplier mechanism. Is hypoxia responsible for the terminal gasp? Can it override the inhibition of hypocapnia? Probably yes, but the evidence is rather slim. The question is relevant in a patient who, like this child, seems to have a totally flat carbon dioxide response. Clearly hypoxia becomes an impotent stimulus if it must be multiplied by zero. But what is it that causes this boy, finally, to breathe twice a minute during sleep? Why not test whether oxygen administration induces total apnea at this point? My guess is that it will, and that we stand to gain a significant insight into the ultimate defense mechanism by testing it.

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